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Linkage mapping of the porcine chromogranin B (*CHGB*) gene to chromosome 17[1]

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Source/description of primers: Chromogranins mediate sorting and aggregation of hormones and neuropeptides into secretory granules in the regulated secretory pathway.[1] Chromogranin B (CHGB)/secretogranin I is found in secretory granules/large dense-cored vesicles throughout cells of the endocrine and nervous system.[2] Because of its role in hormone secretion, CHGB is critically involved in reproductive function. Two cDNA clones, one containing the full coding region (pCHGB1; GenBank accession no. AY613916) and another containing a partial coding region (pCHGB2; GenBank accession no. AY623647) of the porcine *CHGB*, were isolated from the Meat Animal Research Center (MARC) 2PIG expressed sequence tag library.[3] A silent single nucleotide polymorphism (SNP) in the coding region (nucleotide 1568, AY613916; and nucleotide 876, AY623647) was identified by comparing the sequences of the two clones. Primers were designed to amplify a 941 bp product in the coding region of the gene. The forward (F) and reverse (R) primers correspond to bases 1056–1075 and 1996–1975 of the porcine *CHGB* cDNA (GenBank accession no. AY613916).

PCR conditions: In order to further characterize the CHGB gene, PCR reactions were carried out in a 25-μl volume containing 100 ng genomic DNA, 1.5 mM $MgCl_2$, 20 pmol of each primer (*CHGB* F and R), 100 μM dNTP and 0.35 U Hotstar™ *Taq* polymerase (Qiagen Inc., Valencia, CA, USA). Amplification was performed under the following PCR conditions; 15 min at 94 °C; 40 cycles of 30 s at 94 °C, annealing for 45 s at 59 °C, 90 s at 72 °C; and a final extension of 5 min at 72 °C. Both strands of the amplified genomic DNA of parents from the MARC Swine Reference Population[4] were sequenced and evaluated for polymorphisms.[5]

Polymorphism and chromosomal location: The same A/G polymorphism described above (silent SNP at nucleotide 1568 of GenBank accession no. AY613916) was detected in the coding region of *CHGB* and was heterozygous in five of the seven F1 sows and one boar of the MARC Swine Reference Population.[4] An assay was designed to genotype this polymorphism by primer extension with the *CHGB* probe primer and analyte detection on a MALDI-TOF mass spectrometer using a pair of internal primers. The internal forward (*CHGB* Fi) and tailed reverse (*CHGB* Ri) primers correspond to bases 1459–1480 and 1660–1639, respectively, of the porcine *CHGB* cDNA (GenBank accession no. AY613916). This marker generated 64 informative meioses in the MARC swine reference population. The *CHGB* gene was mapped using CRI-MAP to chromosome 17, relative position 30.2 cM, which is the same position as microsatellite marker *SWR1133* on the current MARC swine chromosome 17 linkage map (http://www.marc.usda.gov/). The most significant two-point linkage of *CHGB* detected was with *SWR1133* (LOD = 18.06) at 0.0 recombination. The *CHGB* gene is located on human chromosome 20p12.3, which shares homology with swine chromosome 17.

PCR primer sequences and flanking sequence of a single nucleotide polymorphism: *CHGB* F: CAGAGAACCCGCCATCCTAC
CHGB R: GCCCTCTCCTTCTCACTCTCTG
CHGB Fi: AGGCCAAGCTGAGAAATTACCT
CHGB Ri: GGTGGGACGTAGTATGGATTGA
Sequence flanking polymorphism at nucleotide 1568: AGCACGCTGC(A/G)GAAGCCAGGC
CHGB probe primer: CCATGGGGAGCACGCTGC

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[1]Mention of trade names or commercial products in this article is solely for the purpose of providing specific information and does not imply recommendation or endorsement by the US Department of Agriculture.

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Analysis of the canine *EDAR* gene and exclusion as a candidate for the hairless phenotype in the Chinese Crested dog

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Source/description: Ectodermal dysplasias (ED) are syndromes affecting several organs that develop from the embryonic ectoderm. The most common form of ED in human is hypohidrotic ectodermal dysplasia, also called ED1, EDA or HED.[1] Deleterious mutations in the X-chromosomal ectodysplasin A gene (*EDA*) result in sparse hair on the scalp and body, heat intolerance because of the absence of sweat glands, and abnormal spiky or absent teeth. Similar symptoms are seen in